Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	431	handler.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L2	89	piggybac	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L3	2	L1 and piggybac	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L4	315649	fluorescent or fluorescence	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L5	22893	heat ADJ shock	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L6	7873	ubiquitin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L7	9404	L5 with promoter	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L8	2971	L6 with promoter	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L9	11128	transposon or transposable	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L10	1152	L7 and L9	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L11	1182	L8 and L9	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L12	940	L10 and L4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19

L13	934	L11 and L4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L14	36	L12 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L15	15	L13 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L16	3383	ITR or "inverted terminal repeat"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L17	5	L14 and L16	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L18	1	L15 and L16	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L19	12	(deletion or deleting) WITH L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L20	19947	drosophila	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L21	1894	L20 and L6	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L22	1755	L21 and promoter	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L23	20	L22 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L24	390	bglII WITH HpaI	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19

L25	1	L24 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L26	1	"internal sequence" WITH L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L27	1	minimum WITH L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/07 12:19
L28	38529	(435/320.1|536/23.1|536/24.1|536/24.2|.ccls.)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:24
L29	1	l28 and l1	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:33
L30	24	l28 and l2 and l4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:33
L31	2	l28 and l6 and l4 and l2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:33
L32	182	piggyback.clm.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:34
L33	0	l32 and fluorescent	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:34
L34	25633	fluorescent.clm.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:34
L35	0	l32 and l34	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:34
L36	12	heat-shock.clm. WITH promoter	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:34

L37	0	l36 and l2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:35
L38	77	polyubiquitin NEAR5 promoter	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:35
L39	2	l38 and l2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/10/07 12:35

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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 12:37:02 ON 07 OCT 2005
L1         35273 S HANDLER?/AU OR BEAM?/AU OR HUA-VAN?/AU OR LI-XU?/AU OR FRASER
L2           182 S PIGGYBAC
L3         25961 S TRANSPOSON
L4             5 S (MINIMUM OR INTERNAL) (S) L2
L5            51 S L1 AND L2
L6        693187 S FLUORESCENT OR FLUORESCENCE
L7         82283 S HEAT (2W) SHOCK
L8           309 S UBIQUITIN (2W) PROMOTER
L9        160837 S DROSOPHILA
L10          108 S BGLII (S) HPAI
L11           51 S L1 AND L2
L12           25 DUP REM L11 (26 DUPLICATES REMOVED)
L13           38 S L2 AND L3 AND L6
L14            8 S L13 AND L7
L15            5 DUP REM L14 (3 DUPLICATES REMOVED)
L16           51 S L1 AND L2
L17           17 S L16 AND L9
L18            8 DUP REM L17 (9 DUPLICATES REMOVED)
L19            0 S L10 AND L7 AND L6
L20            0 S L10 AND L1 AND L2
L21           17 S L9 AND L5
L22            8 DUP REM L21 (9 DUPLICATES REMOVED)
L23            0 S L1 AND L2 AND L3 AND L6 AND L8
L24            0 S L8 AND L5
L25            0 S L8 AND L2
L26            6 S L8 AND L9
L27            2 DUP REM L26 (4 DUPLICATES REMOVED)

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L27 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001337995 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11227797
TITLE: Genetic analysis of the requirements for alpha-actinin function.
AUTHOR: Dubreuil R R; Wang P
CORPORATE SOURCE: Department of Neurobiology, Pharmacology, and Physiology, University of Chicago, IL 60637, USA.. ron@drugs.bsd.uchicago.edu
CONTRACT NUMBER: GM49301 (NIGMS)
SOURCE: Journal of muscle research and cell motility, (2000) 21 (7) 705-13.
Journal code: 8006298. ISSN: 0142-4319.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010618
Last Updated on STN: 20010618
Entered Medline: 20010614

AB Null alpha-actinin mutations in **Drosophila** are lethal and produce conspicuous defects in muscle structure and function. Here, we used transgene rescue to examine the requirements for alpha-actinin function in vivo. First, we tested the ability of a cDNA-based transgene encoding the adult muscle isoform of alpha-actinin under control of the heterologous **ubiquitin promoter** to rescue the lethality of null alpha-actinin mutations. Successful rescue indicated that alternative splicing, which also generates larval muscle and non-muscle isoforms, was not essential for viability and that there were no strict spatial or temporal requirements for alpha-actinin expression. Secondly, chimeric transgenes, with functional domains of alpha-actinin replaced by similar domains from spectrin, were tested for their ability to rescue alpha-actinin mutants. Replacement of either the actin binding domain or the EF hand calcium binding domain yielded inactive proteins, indicating that these conserved domains were not functionally equivalent. Thirdly, the length of alpha-actinin was modified by adding a 114 amino acid structural repeat from alpha-spectrin to the center of the rod domain of alpha-actinin. Addition of this sequence module was expected to increase the length of the native alpha-actinin molecule by at least 15%. yet was fully compatible with alpha-actinin function as measured by rescued lethality and flight. Thus, unexpectedly, the exact length of alpha-actinin was not critical to its function in the muscle Z disk.

L27 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 94103334 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8276898
TITLE: Cell shape and interaction defects in alpha-spectrin mutants of **Drosophila** melanogaster.
AUTHOR: Lee J K; Coyne R S; Dubreuil R R; Goldstein L S; Branton D
CORPORATE SOURCE: Department of Cellular and Developmental Biology, Harvard University, Cambridge, Massachusetts 02138.
CONTRACT NUMBER: GM 39686 (NIGMS)
SOURCE: Journal of cell biology, (1993 Dec) 123 (6 Pt 2) 1797-809.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199402
ENTRY DATE: Entered STN: 19940218
Last Updated on STN: 19940218
Entered Medline: 19940204

AB We show that the alpha-spectrin gene is essential for larval survival and development by characterizing several alpha-spectrin mutations in **Drosophila**. P-element minigene rescue and sequence analysis were used to identify the alpha-spectrin gene as the l(3)dre3 complementation group of the Dras-Roughened-ecdysoneless region of chromosome 3 (Sliter et

al., 1988). Germ line transformants carrying an alpha-spectrin cDNA, whose expression is driven by the **ubiquitin promoter,** fully rescued the first to second instar lethality characteristic of the l(3)dre3 alleles. The molecular defects in two gamma-ray-induced alleles were identified. One of these mutations, which resulted in second instar lethality, contained a 73-bp deletion in alpha-spectrin segment 22 (starting at amino acid residue 2312), producing a premature stop codon between the two EF hands found in this segment. The second mutation, which resulted in first instar lethality, contained a 20 base pair deletion in the middle of segment 1 (at amino acid residue 92), resulting in a premature stop codon. Examination of the spectrin-deficient larvae revealed a loss of contact between epithelial cells of the gut and disruption of cell-substratum interactions. The most pronounced morphological change was seen in tissues of complex cellular architecture such as the middle midgut where a loss of cell contact between cup-shaped cuprophilic cells and neighboring interstitial cells was accompanied by disorganization of the cuprophilic cell brush borders. Our examination of spectrin deficient larvae suggests that an important role of non-erythroid spectrin is to stabilize cell to cell interactions that are critical for the maintenance of cell shape and subcellular organization within tissues.

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PALM INTRANET

Day : Friday
Date: 10/7/2005

Time: 12:41:21

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
handler	a	Search

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